IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF ILLINOIS
EASTERN DIVISION

IN RE: ABBOTT LABORATORIES, *et al.*, PRETERM INFANT NUTRITION PRODUCTS LIABILITY LITIGATION	MDL No. 3026
	Master Docket No. 1:22 CV 00071
This Document Relates to:	Judge Rebecca R. Pallmeyer
Diggs v. Abbott Laboratories, Case No. 1:22-CV-05356	

PLAINTIFF'S OMNIBUS MOTION TO EXCLUDE CERTAIN PROPOSED EXPERT TESTIMONY

Under Fed. R. Evid. 702, Plaintiff Keosha Diggs respectfully moves to exclude the testimony of Abbott's retained experts, Drs. Camilia Martin, Brian Smith, and Alex Williamson.

I. Legal Standard

Fed. R. Evid. 702 allows a court to admit expert testimony only when four conditions are met:

> (a) the expert's scientific, technical, or otherwise specialized knowledge will help the trier of fact to understand the evidence or to determine a fact in issue; (b) the testimony is based on sufficient facts or data; (c) the testimony is the product of reliable principles and methods; and (d) the expert has reliably applied the principles and methods to the facts of the case.

Brown v. City of Chicago, No. 18 C 7064, 2023 WL 2561728, at *1 (N.D. Ill. Mar. 17, 2023). In making this determination, courts engage in a three-step analysis which asks "whether the witness is qualified; whether the expert's methodology is scientifically reliable; and whether the testimony will assist the trier of fact to understand the evidence or to determine a fact in issue." *In re Zimmer Nexgen Knee Implant Prods. Liab. Litig.*, No. 11 C 5468, 2015 WL 5050214, at *2 (N.D. Ill. Aug. 25, 2015) (internal quotations and citations omitted). The proponent of the expert testimony bears the burden of showing that the testimony satisfies that standard by a preponderance of the evidence. *Gopalratnam v. Hewlett-Packard Co.*, 877 F.3d 771, 782 (7th Cir. 2017).

II. Law and Argument

A. The Court should exclude Dr. Martin, Abbott's expert on specific causation.

1. Dr. Martin's analysis does not fit the facts of the case.

Dr. Martin opined on whether formula caused NEC in Kamari's case, concluding that "[t]he formula products administered to Kamari did not cause his NEC." *See* Martin Report, Sletvold Decl. Ex. 1, at 35–42. In reaching this conclusion, Dr. Martin relied on her general causation opinion, which included an epidemiological literature review.[1] *Id.* at 41–42. Using that literature review, she reached the conclusion that "(1) high levels of human milk decrease the incidence of NEC and (2) when the

[1] Martin's review of the literature—used for a "dose-response analysis"—is laid out in a chart of studies, the majority of which use cow's-milk-based fortifier with human milk as a comparison to unfortified human milk.

[infant's] diet contains a certain threshold level of human milk, there is no longer an association between formula and NEC." *Id.* at 22. Dr. Martin defines that "threshold level" as 75% human milk:

> Q. Okay. What is that certain threshold that you reference on page 22, Doctor?
> A. Looking at – looking at the studies and the analysis around this, I have – I have testified before that it is around 75 percent.
> Q. Around 75 percent?
> A. Correct.
> Q. Okay. Is it 78 percent? Is it 72 percent? I think the judge and the jury is entitled to know where that kind of around is, and why it's around.
> A. Sure. I would say 70 – 72 to 78; in that range.

Martin Tr. (vol. 3) (Feb. 12, 2025), Sletvold Decl. Ex. 2, at 43:1–19 (objection omitted). This means that if an infant's diet was more than 25% formula, Dr. Martin's analysis does not apply.

Kamari was fed at least 90% formula—a fact Dr. Martin does not dispute. *Id.* at 36:6–8. Dr. Martin conceded that her dose-response opinion would be "wholly inapplicable" to Kamari's case:

> Q. In this case, Kamari had probably 90 to 99 percent formula, correct?
> A. Yes.
> Q. And in fact, he – okay. So I just want to be clear. All of your opinions in your general causation portion of the Diggs report related to the 75 percent of human milk kind of dose opinion that you gave, would be wholly inapplicable to Kamari Diggs, yes?
> A. Right. Right.

Id. at 36:6–17. This makes her opinion irrelevant to this case.

An expert's opinion must be excluded if it does not fit the facts of the case. *See Porter v. Whitehall Lab'ys, Inc.*, 9 F.3d 607, 616 (7th Cir. 1993); *In re Testosterone Replacement Therapy Prods. Liab. Litig. Coordinated Pretrial Proceedings*, No. 14 C 1748, MDL No. 2545, 2017 WL 4772759, at *7 (N.D. Ill. Oct. 23, 2017). *In re TRT* is instructive. There, the defendant's expert was excluded because "his proposed testimony [did] not fit the facts of [the plaintiff's] case." *Id.* The expert based his specific-causation opinion on the premise that the plaintiff applied the drug in question as prescribed and used the full recommended dose. *Id.* The expert testified that he had no opinion as to whether the drug could cause injury when misapplied or when using less than the recommended dose. *Id.* But the evidence made

clear that the plaintiff had *not* applied the drug as instructed or used the full dose. *Id.* Because of the lack of fit, the court excluded the expert's opinion.

Dr. Martin's opinion suffers from almost identical faults. Dr. Martin has unambiguously testified that her analysis—which is premised upon an infant being fed between 72% and 78% human milk—does not apply to the facts of the Diggs case. Ex. 2 (Martin Tr.) at 36:6–17. Much like the expert in *TRT*, Dr. Martin's opinion can only be relevant to other plaintiffs, and even then only in certain circumstances. Her specific-causation opinion should be excluded.

2. Dr. Martin should be excluded because her methodology is unreliable.

Even if Dr. Martin's analysis did fit the facts of this case, it should still be excluded because it is the product of an unreliable methodology. In evaluating whether formula-feeding was a cause of Kamari's NEC, Dr. Martin relied on her general-causation opinion, asserting that "the presence of preterm formula does not cause or contribute to the development of NEC. It therefore could not have caused or contributed to the development of Kamari's NEC." Ex. 1 (Martin Rep.) at 41–42. But the general-causation opinion upon which Dr. Martin relies suffers from fatal methodological flaws.

i. Dr. Martin used an unreliable methodology in her systematic literature review, which lacks support.

Dr. Martin's cites three bases for her opinion that the epidemiological literature does not show a causal relationship between formula and NEC: (1) systematic review, (2) limitations of meta-analyses, and (3) Bradford Hill. Ex. 1 (Martin Rep.) at 19–24. None of these arguments are based on reliable or sound methodological principles.

The faults in Dr. Martin's methodology are abundant. First, as support for her conclusion that formula does not cause NEC, Dr. Martin cites the NIH's funding of over $100 million in NEC research and that no epidemiological study has concluded that preterm products cause NEC. *Id.* at 19. But it is well-established that many medical journals "restrict the use of causal language to the reporting of randomized clinical trials." *See* Case No. 1:22-cv-00071, ECF No. 614-46 (Dahabreh, Issa J. et al.

Causal Inference About the Effects of Interventions from Observational Studies in Medical Journals. JAMA. 2024; 331(21):1845-1853. Doi:10.1001/jama.2024.7741). This means that a publication may not explicitly endorse a causal relationship not because one does not exist, but because the journal in which it is published does not allow such statements. So, contrary to well-established principles in medical and scientific research, Dr. Martin improperly relies on the absence of causal language as supportive of her opinion. This is not consistent with the methodology upon which experts in the field would rely, as required by Rule 702.

Dr. Martin then argues that scientific literature shows that when premature infants receive "a small proportion" of human milk in their diet, NEC incidence "may be higher, but this is due to the low levels of protective components and not anything harmful or causal in formula." Ex. 1 (Martin Rep.) at 20. The only support for this *ipse dixit* conclusion is Dr. Martin's "dose-response analysis"—the same one that does not fit the facts of this case because it depends on a 72 to 78% "threshold level" of human milk. *See supra*. Beyond its irrelevance to Kamari's case, the dose-response analysis suffers from methodological problems. It does not include any guidance or quantitative analysis regarding what the "protective effect" of human milk is. Dr. Martin even confirmed in her deposition that she cannot quantify the protection of human milk:

> Q. Doctor, so let me give you a hypothetical. If a baby is given human milk for 30 days, and then switches to formula, and gets to full volume formula feeds within five days after that switch, 30 to 35 days; are you with me so far?
> A. Yes.
> Q. How much protective effect did those 30 days of human milk give that baby?
> A. I don't think it's ever been quantified like that. Knowing the biology and physiology of what human milk does, I would have – I would assume a fair amount of protection.
> Q. You would assume it, but you don't know, right?
> A. Well, I don't think you can do those studies exactly.
> Q. Right.
> A. But based on the biology and physiology as we know it, and what exposure to human milk does, 30 days, straight days of human milk and then formula, that's a lot of good protection for – with the human milk.
> Q. Well, when you say a lot of protection, and these assumptions you make, how much protection?

A. I don't think studies can delineate that to that degree.
Q. So it is hard to quantify?
A. I would say so.

Ex. 2 (Martin Tr.) at 82:1–83:7. Dr. Martin cannot quantify how much protection human milk provides an infant and does not cite any data to support her conclusory opinion that any increased incidence of NEC "is due to the low levels of protective components and not anything harmful or causal in formula." Ex. 1 (Martin Rep.) at 19. Dr. Martin's unsupported dose-response analysis should be excluded.

Dr. Martin's analysis generally lacks support, but that becomes even more problematic after eliminating the sources she cites but cannot use in this litigation. First, Dr. Martin relies on a so-called "Consensus Statement" by the FDA, NIH, and CDC.[2] Ex. 1 (Martin Rep.) at 22. The Court already precluded the use of this press release in this litigation. Transcript of Proceedings – Pretrial Conference in Case No. 1:22-cv-00232 (Apr. 17, 2025), Sletvold Decl. Ex. 3, at 62:11–24. Therefore, any part of Dr. Martin's report that relies upon the press release should be excluded.

Dr. Martin also argues that "historical trends in human data" do not support causation. Ex. 1 (Martin rep.) at 22. To support that proposition, Dr. Martin cites to the Vermont Oxford Network ("VON") database. This is a misuse of VON's proprietary data. As an initial matter, only members are permitted to use VON data. It is unclear whether Dr. Martin is a member herself, she used the membership of an institution with which she is affiliated, or she accessed VON data from public

[2] "But if formula caused NEC (in addition to human milk protecting against it), there would be two variables, both driving in the same direction, which should lead to a much more consistent and stronger association. This supports the scientific consensus—as expressed by the Consensus Statement joined by the FDA, CDC, and NIH—attributes the associations we see in the literature to the protective components in human milk. If formula was causing NEC (in addition to human milk protecting against it), we would see a stronger association." Ex. 1 (Martin rep.) at 22.

websites.[3] Regardless of how Dr. Martin accessed the data, VON's Policy on Data Use specifically prohibits Dr. Martin's use of the data in her report:

> In no event shall any member use VON Data, in whole or in part, for any of the following purposes: … 3. To make presentations or publish results outside of the member's institution[.]

Vermont Oxford Network Policy on Data Use, Sletvold Decl. Ex. 5, at 2. In other words, no VON member may use VON data outside of the member's own institution. Even in the unlikely event that Dr. Martin used VON data in relation to her own research, the use still violates VON's Policy and Guidelines for Collaborative Research Using the Vermont Oxford Network Databases. Vermont Oxford Network Policy and Guidelines on Collaborative Research Using the Vermont Oxford Network Databases, Sletvold Decl. Ex. 6. Even assuming Dr. Martin followed the procedures outlined by VON for collaborative research, including submitting a proposal to VON and including VON staff members in the research process, Dr. Martin's research is not "free of commercial bias or conflict of interest." *Id.* at 3.

Even if Dr. Martin's use of VON data complied with VON's requirements, it would be unfair to allow Dr. Martin's use of the data in her expert opinion. Ms. Diggs is not a member of VON and cannot access and use the data in compliance with VON's policies and user agreements, so she is prohibited from evaluating the misused data upon which Dr. Martin relies. *See EEOC v. Hillstone Restaurant Group, Inc.*, No. 22-CV-3108 (JRL) (RWL), 2025 WL 1285740, at *1 (S.D.N.Y. May 2, 2025) (requiring subpoena for third-party data because the terms of use would prohibit the defendant from using the data but the defendant needed it to "independently investigate the accuracy of the DOB/age data on which EEOC will rely"). Ms. Diggs cannot be forced to violate VON's use policies to defend

[3] Dr. Martin testified that she obtained the data through VON's "publicacly [*sic*] available website." Martin Tr. (vol. 1) (Feb. 11, 2025), Sletvold Decl. as Ex. 4, at 389:10-21). Ms. Diggs was unable to access the databases through VON's public website without membership credentials. https://public.vtoxford.org/. Further, Dr. Martin's Third Amended Reliance List, items 432–449, reference VON database summaries, which Ms. Diggs cannot obtain or access. *See* Third Amended Reliance List of Dr. Camilia Martin, Sletvold Decl. as Ex. 7.

against Dr. Martin's reliance on data that was improperly used in the first place. References to VON sources should be excluded.

After removing the press release and VON data, neither of which can serve as the basis for her opinion, Dr. Martin's questionably supported analysis becomes glaringly lacking in support. It is pure *ipse dixit* and should be excluded.

ii. Dr. Martin's lack of a meta-analysis makes her epidemiological review incomplete and unreliable.

Moving past the methodological issues with Dr. Martin's systematic literature review and corresponding dose-response analysis, more issues arise from her lack of a meta-analysis. Although meta-analysis is a reliable and widely accepted methodology, Dr. Martin did not conduct a meta-analysis, claiming that "the epidemiological literature and data do not readily lend themselves to a meta-analysis" and noting the variability in randomized controlled studies ("RCTs"). Ex. 1 (Martin Rep.) at 23. Instead, Dr. Martin created her own methodology: combining RCTs to support her opinion regarding the threshold use of 75/25% dose response for finding that there is nothing harmful or causal in formula. Dr. Martin's own arbitrary and unsupported methodology replaced the "time-honored approach of reviewing the literature, which is characteristic of science, and placing it in a standardized framework with quantitative methods for estimating risk." *See Fed. Jud. Ctr.* Reference Manual on Scientific Evidence ("RMSE") at 607 (3d ed. 2011).

Then Dr. Martin took a step even further from the well-established scientific methodology of a meta-analysis by completely ignoring observational studies. Dr. Martin noted that observational studies should not be combined with RCTs in a meta-analysis, but did not analyze observational studies independently of RCTs either. Ex. 1 (Martin Rep.) at 23. Instead, she summarily discounted them as "less reliable" and excluded them from the general-causation analysis upon which she based her specific-causation analysis. *Id.* at 24. This is not only inconsistent with standard methodological principles of epidemiology, but is also inconsistent with Dr. Martin's stated methodology. *Id.* at 6

(stating she considered cohort studies, case-control studies, and case reports). An expert's own methodology (which she does not follow to the letter anyway) is not a reliable basis for a conclusion admissible under Rule 702.

iii. Dr. Martin's analysis of Bradford Hill factors is unsupported and unreliable.

Finally, Dr. Martin addresses a Bradford Hill analysis. This section of her report predominantly consists of Dr. Martin lodging her disagreement with the Bradford Hill analysis of Plaintiff's epidemiologist Dr. Spector. *Id.* at 23–24. Dr. Martin does not cite a single source in support of her disagreement with any Bradford Hill factor, save one. The lone Bradford Hill factor that Dr. Martin addresses with support is temporality. She disputes Dr. Spector's assertion that "it stands to reason" that the studies, by design, involved preterm infants who were fed preterm formula prior to developing NEC. *Id.* at 24. She cites a study to support her proposition that there is variability in lengths of time between exposure and NEC. *Id.* Ms. Diggs does not disagree with that proposition, but it is disconnected from Dr. Martin's conclusion that it "removes temporality as a consistently met criterion of Bradford Hill analysis." *Id.* Time between exposure to formula and NEC can vary; this does not change the fact that exposure to formula preceded a NEC diagnosis in the studies Dr. Spector used in his analysis.

This Bradford Hill analysis is nothing but more *ipse dixit* from Dr. Martin. If anything, it is solely an attempt to discredit Dr. Spector (who has already survived the Rule 702 phase of this litigation) rather than to bolster Dr. Martin's own opinion. It should be excluded.

B. The Court should exclude Dr. Smith, Abbott's other expert on specific causation.

1. Dr. Smith's analysis is based on a faulty methodology.

Dr. Smith's case-specific analysis and conclusions were based on a review of Kamari's and Ms. Diggs's medical records and of certain scientific literature. Smith Report, Sletvold Decl. Ex. 8, at 6. But Dr. Smith's conclusion about whether formula caused Kamari to contract NEC was based entirely

on his general-causation opinion. *Id.* at 14. He concluded that, "as outlined in [his] opinions above, preterm infant formula products do not cause or substantially contribute to the development of NEC in premature infants." *Id.* And those opinions are unreliable because they are based on a flawed analysis.

Dr. Smith's analysis was faulty for two reasons. First, he built his methodology using that of the Working Group on NEC, which explicitly did not reach a conclusion on causation. Second, he excluded numerous relevant studies from his analysis.

i. Dr. Smith based his "methodology" on that of the Working Group on NEC, which did not analyze causation.

Dr. Smith's methodology was conceived from another group's methodology that was not intended to reach a causal conclusion. Dr. Smith's "reliance on the overall strength and direction of association and consistency of the scientific literature is also consistent with the approach taken by the Department of Health and Human Services NACHHD Working Group on NEC in Preterm Infants." Ex. 8 (Smith Rep.) at 6. The Working Group explicitly stated in its report, however, that its "charge and timeframe did not allow for the application of frameworks to determination [*sic*] causation versus association, or to systematically evaluate the certainty of evidence." ECF No. 605-2, PageID #14345. Employing the same methodology elsewhere can only lead to the same result—an inability to differentiate between causation and association.

Dr. Smith's adoption of the Working Group's methodology is suspect enough, but he goes on to rely on the contents of the Working Group report to deny causation:

> The Department of Health and Human Services NACHHD Working Group on NEC in Preterm Infants recently recognized this, concluding that "[a]vailable evidence supports the hypothesis that it is the absence of any human milk in an infant's diet that is associated with a higher risk of NEC." This report explained that "much of the current evidence shows only associations, not direct or indirect causation linking biological pathways to risk factors for development and/or progression of NEC" and that "[s]tudies showing an association between a risk factor and the development of NEC do not necessarily mean that the factor causes NEC."

Ex. 8 (Smith Rep.) at 10. In other words, Dr. Smith represented the Working Group report as conclusive evidence that formula does not cause NEC. Allowing such testimony would not only give the false impression that the Working Group reached a conclusion regarding causation, which it expressly said it did not, but would also violate the Court's order precluding statements describing the Working Group report as "conclusive." Ex. 3 (Pretrial Hearing Tr.) at 62:11–24.

But that is not the only part of Dr. Smith's report that violates the Court's ruling. Dr. Smith also uses the so-called "consensus statement," which the Court excluded, to bolster his argument:

> This conclusion was recently reaffirmed by a joint NIH, FDA, and CDC consensus statement: "There are two key points about feeding practices and NEC: 1) There is no conclusive evidence that preterm infant formula causes NEC; and 2) there is strong evidence that human milk is protective against NEC. Available evidence supports the hypothesis that it is the absence of human milk—rather than the exposure to formula—that is associated with an increase in the risk of NEC."

Ex. 8 (Smith Rep.) at 10. The press release (masquerading as a consensus statement) has been excluded by the Court because it is misleading, suggesting that the Working Group reached a definitive conclusion that formula does not cause NEC, when the Working Group itself makes clear that it did not reach such a conclusion. Dr. Smith's testimony regarding the press release should be excluded—but his reliance on the Working Group for methodological and substantive guidance also supports excluding his opinion.

ii. Dr. Smith excluded studies that were not randomized controlled trials even if they were relevant to his analysis.

Dr. Smith's methodology included a literature review. After conducting an initial search for literature using specific search terms,[4] Dr. Smith narrowed down the pool of studies to review for his report by reading each study's title and abstract. Ex. 8 (Smith Rep.) at 5. He "identif[ied] studies that appeared to meet [his] predefined eligibility criteria[,]" which included:

[4] Dr. Smith's search terms were limited to "Breast milk," "Prolacta," "Donor Breast Milk," "Necrotizing Enterocolitis" OR "Necrotising Enterocolitis," and "Medolac." Curiously, his search terms did not include any words or phrases related to cow's-milk-based formula. Ex. 8 (Smith rep.) at 5.

- Patient Population: Premature (<37 weeks completed gestational age) or very low birth-weight infants (birthweight <1500 g).
- Intervention: Primarily human milk or human milk product.
- Control: Diet containing some portion of bovine derived preterm formula and fortifier products.
- Outcomes: NEC (primary or secondary outcomes).
- Study Design: Randomized controlled trials (RCT).

Id. This list of criteria automatically excluded studies that were not randomized controlled trials even when they contained relevant information for his analysis.

When an area of scientific study has lost equipoise, it is no longer ethical to conduct RCTs. *See In re Abbott Laby's, et al., Preterm Infant Nutrition Prods. Liab. Litig.*, No. 22 C 00071, MDL No. 3026, 2025 WL 1283927, at *2 n.3 (N.D. Ill. May 2, 2025) ("The study observes the difference in outcomes between the two groups of participants, and any RCT must end (for ethical reasons) the moment researchers have reason to suspect that exposure to the agent is harmful."). Where there is a lack of equipoise, other types of studies may be ethically conducted instead—namely, observational studies such as cohort studies and case-control studies. *Id.* These studies can be just as useful as RCTs in gathering information about a study topic.

Excluding non-RCTs from a literature review eliminates valuable data that can help inform an expert's opinion. Dr. Smith's automatic exclusion of *all* non-RCTs from his primary analysis prevented him from reviewing numerous relevant, useful studies that his search returned, including:

- Richard J. Schanler, et al., *Feeding Strategies for Premature Infants: Beneficial Outcomes of Feeding Fortified Human Milk Versus Preterm Formula*, 103 Pediatrics 1150 (1999) ("The incidence of necrotizing enterocolitis and late-onset sepsis was less in the FHM group."), Sletvold Decl. Ex. 9.
- Amy B. Hair, et al., *Beyond Necrotizing Enterocolitis Prevention: Improving Outcomes with an Exclusive Human Milk-Based Diet*, 11 Breastfeeding Med. 70 (2016) ("The HUM group had significantly lower incidence of proven NEC (16.7% versus 6.9%, $p < 0.00001$), mortality (17.2% versus 13.6%, $p = 0.04$), late-onset sepsis (30.3% versus 19.0%, $p < 0.00001$), ROP (9% versus 5.2%, $p = 0.003$), and BPD (56.3% versus 47.7%, $p = 0.0015$) compared with the BOV group."), Sletvold Decl. Ex. 10.
- Syed M. Assad, et al., *Decreased cost and improved feeding tolerance in VLBW infants fed an exclusive human milk diet*, 36 J. Perinatology 216 (2016) ("Feeding intolerance occurred

less often ($P<0.0001$), number of days to full feeds was lower ($P<0.001$), incidence of NEC was lower ($P<0.011$), and total hospitalization costs were lower by up to $106,968 per infant ($P<0.004$) in those fed an EHM diet compared with the other groups."), Sletvold Decl. Ex. 11.

- Juliane Spiegler, et al., *Does Breastmilk Influence the Development of Bronchopulmonary Dysplasia?*, 169 J. Pediatrics 76 (2016) ("Exclusive formula feeding of very low birth weight infants was associated with increased risks of BPD (OR 2.6) as well as NEC (OR 12.6) and ROP (OR 1.80) after controlling for known risk factors."), Sletvold Decl. Ex. 12.
- Paula M. Sisk, et al., *Necrotizing Enterocolitis and Growth in Preterm Infants Fed Predominantly Maternal Milk, Pasteurized Donor Milk, or Preterm Formula: A Retrospective Study*, 34 Am. J. Perinatology 676 (2017) ("NEC rates were different by feeding group (MM: 5.3%; PHDM: 4.3%; PF: 11.4%; $p = 0.04$)."), Sletvold Decl. Ex. 13.
- Wenjing Peng, et al., *The Association of Human Milk Feeding With Short-Term Health Outcomes Among Chinese Very/Extremely Low Birth Weight Infants*, 38 J. Human Lactation 670 (2022) ("Exclusive human milk feeding was associated with lower odds of necrotizing enterocolitis (2.90% vs. 8.42%, OR 0.33, 95% CI [0.22, 0.47]… compared with formula feeding."), Sletvold Decl. Ex. 14.

See generally Ex. 8 (Smith Rep.). Each of these non-RCTs measured NEC as a study outcome, showed that human milk is associated with lower rates of NEC, and included that information in the abstract, meaning it would have been included if Dr. Smith's methodology did not wantonly exclude non-RCTs. Those studies' inclusion likely would have changed the outcome.

Those studies were excluded from Dr. Smith's primary analysis, but were included in his secondary analysis. *Id.* at B-1–B-5. But he *never* analyzed other relevant studies that his search returned, such as:

- Daniel J.C. Berkhout, et al., *Risk Factors for Necrotizing Enterocolitis: A Prospective Multicenter Case-Control Study*, 114 Neonatology 277 (2018) ("Formula feeding and prolonged (duration of) parenteral feeding were associated with an increased risk of NEC."), Sletvold Decl. Ex. 15.
- Aleksandra Kaplina, et al., *Necrotizing Enterocolitis: The Role of Hypoxia, Gut Microbiome, and Microbial Metabolites*, 24 Int. J. of Molecular Sciences 2471 (2023) ("Breast milk administration and probiotics are known to effectively protect against NEC, while abnormal bacterial colonization and formula feeding are major postnatal factors contributing to NEC, apart from prematurity."), Sletvold Decl. Ex. 16.
- Maria Quigley, et al., *Formula versus donor breast milk for feeding preterm or low birth weight infants*, Cochrane Database Sys. Review (2019) ("Formula feeding increased the risk of necrotising enterocolitis (typical risk ratio (RR) 1.87, 95% CI 1.23 to 2.85; risk difference (RD) 0.03, 95% CI 0.01 to 0.05; number needed to treat for an additional

> harmful outcome (NNTH) 33, 95% CI 20 to 100; 9 studies, 1675 infants)."), Sletvold Decl. Ex. 17.

See generally Ex. 8 (Smith Rep.). Dr. Smith's failure to even consider these studies skews his methodology even further in favor of Abbott's position for no legitimate methodological reason—these studies meet the same criteria as the non-RCTs he included in his secondary analysis. This flawed methodology shows that Dr. Smith's opinions are unreliable and his testimony should be excluded.

2. Dr. Smith should be excluded because his testimony is duplicative of Dr. Martin's testimony.

Dr. Smith's testimony is not only unreliable, but duplicative. Both Dr. Smith and Dr. Martin opine on specific causation and, unsurprisingly, both reach the conclusion that Abbott wanted—that Kamari's NEC was not caused by formula because formula does not cause NEC, but human milk protects against it. The two even phrased their conclusions similarly, with Dr. Smith saying:

> Feeding with bovine-derived preterm infant formula is often described as a risk factor for NEC due to the association described above. However, there is an important difference between associations and causation. In fact, as outlined in my opinions above, preterm infant formula products do not cause or substantially contribute to the development of NEC in premature infants. Instead, it is the absence of human milk—especially mother's own milk—that explains the associations shown in some studies between bovine-derived preterm infant formula products and NEC. As explained above, it is not possible to infer a causal relationship between bovine-derived preterm infant formula products and NEC in premature infants generally, and thus, I cannot attribute Kamari's specific case of NEC to formula. Further, Kamari received almost exclusively formula for over a week. During this week, he had no issues with feeding tolerance—he did not have significant residuals, did not have emesis, abnormal stooling, nor abdominal distention, and was growing well. In addition, given Kamari's gestational age and birthweight, he would have been excluded from almost all of the randomized trials and cohort studies that examine the association of bovine derived products and NEC because he did not meet the eligibility criteria. Therefore, the randomized controlled studies, observational studies, and meta-analyses or epidemiological summaries that Plaintiff's experts cite and rely upon do not apply to Kamari.

Id. at 14. Meanwhile, Dr. Martin said:

> Preterm formula has been identified as an associative risk factor for NEC. As discussed above, however, while preterm formula has been associated with NEC, the presence of preterm formula does not cause or contribute to the development of NEC. It therefore could not have caused or contributed to the development of

> Kamari's NEC. In any event, there is no mechanism by which any nutritional composition administered to premature infants in the NICU could cause NEC. Kamari tolerated preterm formula for more than a week before he was diagnosed with NEC—he had no significant aspirates, no abdominal distention, or bloody stools during this time. He was gaining weight as expected, against indicating that he was tolerating and growing while receiving Abbott's preterm formula. Further, given his birthweight and gestational age, he would be excluded from nearly all randomized clinical trials and cohort studies that include NEC as an observed clinical outcome. And, as discussed above, there is no mechanism of action by which preterm infant formula causes NEC and the epidemiological literature does not support that preterm formula causes NEC—particularly as to infants at Kamari's gestational age and birthweight. Therefore, I have ruled out formula as a potential cause of or substantial contributing factor in causing Kamari's NEC.

Ex. 1 (Martin Rep.) at 41–42. These conclusions are suspiciously similar, both saying that Kamari had tolerated formula feeds for a week before developing NEC, that he would be excluded from clinical trials because of his gestational age and birthweight, that there is a difference between association and causation, and more. There is no need for the jury to hear the exact same information from two experts. Dr. Smith's duplicative and needlessly cumulative testimony should be excluded.[5]

C. The Court should exclude Dr. Williamson, Abbott's expert on pathology.

1. Dr. Williamson's testimony should be excluded because it is irrelevant.

Dr. Williamson's testimony should be excluded because it is irrelevant. Evidence can only be helpful to a trier of fact if it is relevant, so an expert's testimony may be excluded where the proponent cannot use it to support any element of the claim at issue. *See, e.g.*, *Jones v. Nat'l Council of Young Men's Chirstian Ass'ns. of the United States of America*, 34 F. Supp. 3d 896, 901 (N.D. Ill. 2014) (excluding an expert's opinions where "they are not relevant to establishing the plaintiffs' disparate impact claims[,]" which were the only claims in the litigation); *Fairley v. Andrews*, No. 03 C 5207, 2011 WL 2142800, at *4 (N.D. Ill. May 31, 2011) ("In other words, there has to be a connection between [the expert]'s expert opinions and the remaining claim in this lawsuit for his opinions to fulfill the helpfulness standard under Rule 702 because '[e]xpert testimony which does not relate to an issue in the case is

[5] In the alternative, Dr. Martin's testimony should be excluded as being duplicative of Dr. Smith's testimony.

not relevant, and ergo, not helpful.'"). Dr. Williamson does not opine on any element of Ms. Diggs's claims, so his testimony should be excluded.

Dr. Williamson's opinion applies strictly to whether Kamari was exposed to an infection in utero. Report of Dr. Alex Williamson, Sletvold Decl. as Exhibit 18, at 6. That fact has no bearing on the resolution of this case—the question at hand is whether Abbott's preterm infant formula caused Kamari to suffer from NEC, not whether he was exposed to infection. And Dr. Williamson does not relate his conclusion about infection back to NEC.

In fact, Dr. Williamson hardly mentions NEC in his report, and when he does, it is in a statement of fact that is not disputed.[6] He took a step even farther from relevance in his deposition, where he testified specifically that his opinion does not go to causation:

> Q. And you don't talk about or address the question of how Kamari came to have necrotizing enterocolitis either?
> A. I did not specifically comment on necrotizing enterocolitis or how he came to have it.
> Q. And you're not addressing and you won't offer an opinion as to the cause of the NEC either in Baby Mar, correct?
> A. Correct, not the cause of necrotizing enterocolitis.
> Q. And not the cause of – of NEC in Kamari Brown; is that right?
> A. Correct. I just – I may address risk factors that may be associated with those outcomes, but not the cause of the outcome in each of those specific patients. So generalized terms, generalized discussions, but not specifically related to Mar or Diggs.

Williamson Tr. (Jan. 10, 2025), Sletvold Decl. Ex. 19, at 134:23–135:19. And that testimony about risk factors is puzzling, as Dr. Williamson proceeded to testify that his report does not discuss risk factors for NEC and that he would not offer an opinion outside the bounds of his report:

> Q. And you don't actually address risk factors in your – in either of these reports?

[6] Dr. Williamson's references to NEC in his report are limited to the following: "I have performed or reviewed dozens of postmortem examinations in the setting of preterm birth, I have performed or reviewed approximately a dozen postmortem examinations involving clinical necrotizing enterocolitis, and I have reviewed nearly two dozen surgical specimens resected from children with clinically suspected necrotizing enterocolitis."; "On February 17, 2015, medical records show that Kamari remained in the NICU and had gas in his gastrointestinal tract that continued to spread throughout his intestine, consistent with a diagnosis of NEC." Ex. 18 (Williamson Rep.) at 2, 4.

A. Not in writing, no. Not – not in either report.
Q. And you do not intend to offer an opinion outside the opinions that you've expressed in these reports?
A. No, not at this time, I do not.

Id. at 135:20–136:4.

In other words, Dr. Williamson offers no opinion about what caused Kamari to develop NEC or what risk factors may have contributed to Kamari's NEC—the issues upon which this case is centered. Dr. Williamson only offers an opinion about whether Kamari was exposed to an infection in utero, and even that he cannot testify to with certainty:

Q. And you cannot say to a reasonable degree of medical certainty that Kamari Brown had an infection while in utero?
A. Correct.

Id. at 14:1–6 (objection omitted). This amounts to nothing more than speculation about an infection to which Kamari might have been exposed. It cannot help the jury reach a conclusion on any of the outstanding issues and therefore should be excluded.

2. Dr. Williamson's testimony should be excluded because it lacks analysis and is therefore unhelpful.

Even if Dr. Williamson's testimony was relevant (it is not), it should be excluded because it is not helpful to the trier of fact due to its lack of analysis. In a products-liability context, a proffered expert opinion that "offers no analysis or discussion of any particular element of [a] plaintiff's complex medical history" is inadmissible. *Teran v. Coloplast Corp.*, 633 F. Supp. 3d 1103, 1112 (N.D. Ill. 2022). An expert's testimony may also be inadmissible if the expert "offer[s] merely a bottom line, as doing so supplies nothing of value to the judicial process." *Id.* It is "a classic example of expert *ipse dixit*." *Id.*

In *Teran*, the plaintiff's expert offered a one-paragraph statement of his opinions, which were based on the totality of the medical records he reviewed. *Id.* The court held that his opinion was "just [the] type of unhelpful 'bottom line'" that should be excluded under Rule 702. *Id.* Dr. Williamson's opinion is much the same.

Dr. Williamson's opinions are contained within three paragraphs of his 43-page report.[7] They read as follows:

> Amniotic fluid inflammation—which in this case includes chorioamnionitis and cord phlebitis and arteritis—is consistent with infection of the amniotic sac, which commonly accompanies preterm premature rupture of membranes ("PPROM"). In this case, both the mother and fetus demonstrated evidence of an inflammatory response. Chorioamnionitis is often seen as a condition of PPROM when the uterine environment becomes open to the external environment, allowing bacteria to ascend from the vagina, through the cervix, and into the uterine cavity. Ms. Diggs was diagnosed with PPROM before she gave birth to Kamari. The uterine environment should be a sterile place (i.e., without bacteria) for the fetus to grow and develop. In this case, however, the unborn infant's immune system responded to the likely amniotic fluid infection he was exposed to in utero before he was delivered.
>
> The focal accelerated villous maturation probably reflects localized maternal vascular malperfusion, which, depending on its extent, can impact the exchange of nutrients between a mother and her fetus.
>
> Here, my observations and findings of the placental pathology are consistent with Dr. Alexiev's surgical pathology report, indicating that the fetus was likely exposed to infected amniotic fluid.

Ex. 18 (Williamson Rep.) at 6. Not only is this abbreviated opinion based on circumstantial probabilities (*e.g.*, "the unborn infant's immune system responded to the *likely* amniotic fluid infection"), but it is also devoid of analysis. Half of the first paragraph is spent explaining the relationship between PPROM and amniotic fluid inflammation. The other half explains that Ms. Diggs was diagnosed with PPROM before giving birth to Kamari and explains how bacteria in the uterine environment can be harmful, but it does not explain why Dr. Williamson believes this affected Kamari or how he reached that conclusion.

The second paragraph provides that localized maternal vascular malperfusion can impact the exchange of nutrients between mother and unborn child *depending on its extent*. Dr. Williamson does not detail the extent of the condition in Kamari's case or explain the impact on the exchange of

[7] Only five of the 43 pages of the report contain substantive discussion. The other 38 pages consist of a cover page, Dr. Williamson's curriculum vitae, images of the slides Dr. Williamson examined, a list of sources Dr. Williamson considered, and a table of Dr. Williamson's previous experiences testifying.

nutrients that it caused. In other words, Dr. Williamson's opinion is based on generalized statements that cannot be applied directly to Kamari—at least not using only the information from Dr. Williamson's report.

The third paragraph simply states that Dr. Williamson's conclusions are consistent with Kamari's doctor's pathology report, which indicated a likely exposure to infected amniotic fluid. This cannot help the jury—it is simply cumulative evidence, presented to prove a fact that can be proven through more reliable evidence (Keosha Diggs's medical records) and that is not in contention anyway. The evidence should be excluded.

3. Dr. Williamson's testimony should be excluded because it will mislead the jury.

Dr. Williamson's testimony is also inadmissible because it is misleading. An expert's testimony may be excluded where it is speculative and will mislead the jury. *See Calvente v. Ghanem*, No. 20-CV-03366, 2022 WL 4272779, at *9 (N.D. Ill. Sept. 15, 2022) ("It is speculative and misleading to suggest, as Dr. Zaeske's opinion does, that Plaintiff is currently qualified for tenure at UNC or that Plaintiff was qualified for tenure at DePaul in 2019 when DePaul denied her tenure."). That is yet another problem with Dr. Williamson's testimony: he cannot say with the requisite certainty whether Kamari was exposed to an infection in utero. Ex. 19 (Williamson Tr.) at 14:1–6. All he can say is that there was amniotic fluid inflammation, which he cannot definitively attribute to an infection:

> Q. So your review of the placental slides confirmed amniotic fluid inflammation or histological chorioamnionitis. Is that right?
> A. Yes. Amniotic – there's various terms: amniotic sac infection, chorioamnionitis, amniotic fluid inflammation. And I will actually clarify, as a pathologist I diagnose inflammation. Infection's the most common cause of inflammation, but I don't necessarily – I can't tell by looking at this tissue that infection's the cause. That's where clinical correlation comes in. It's the most common cause, but I diagnose inflammation, "itis." So there's amniotic fluid or sac or cavity inflammation, including maternal inflammatory response and fetal inflammatory response.
> Q. And that's all you can diagnose from looking at these slides. Is that right?
> A. Yes, with their stage and their grade. In terms of the inflammation, yes, that's all that was present in this case. I can diagnose a lot of other things, but they're not present in this case.

Id. at 118:24–120:5. This does not prove that there was an infection, let alone that the infection affected Kamari. But it will mislead the jury into believing that a possible undiagnosed infection contributed to Kamari's development of NEC.

Dr. Williamson's testimony is also misleading because its speculation that Kamari was exposed to an infection directly contradicts Kamari's medical records without addressing the inconsistency. When he was first admitted to the NICU, the hospital took a blood culture and started him on antibiotics because they were concerned about a potential infection. Medical Record of Kamari Brown (May 10, 2015) (Minor-KB-UniversityMMC-MD-002426-003054), Sletvold Decl. Ex. 20, at 002446. The records confirm that "[h]is blood culture and CSF cultures were negative." *Id.* In other words, *Kamari did not have an infection*, even if there was inflammation in the placental pathology. This destroys any credibility that Dr. Williamson's report may have had—there was no infection, so exposure to a possible infection that Dr. Williamson cannot confirm anyway could not have affected Kamari.

In addition to being blatantly wrong, Dr. Williamson's testimony is all the more misleading in light of the medical records. The records confirm that Kamari did not have an infection. But Dr. Williamson intends to tell the jury that he believes Kamari *did* have an infection, based on nothing but placental slides and speculation. He did not examine Kamari or test him for infections when Kamari was born. And he makes clear that he can only diagnose inflammation, not infection. Ex. 19 (Williamson Tr.) at 118:24–120:5. Allowing Dr. Williamson to testify to this would improperly lead the jury to believe that Kamari suffered from an infection before he developed NEC and draw the incorrect conclusion that this supposed infection caused Kamari to develop NEC (even though Dr. Williamson explicitly declined to opine on what caused Kamari to get NEC). Abbott has no use for this testimony other than to improperly prod the jury toward this conclusion. The testimony should be excluded.

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Respectfully submitted,

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